CLAIMS

1. A non-human animal having a neurologic disease induced by the process of:

perfusing the non-human animal with a pharmacologically effective amount of a combination of an $A\beta$ compound, at least one pro-oxidative compound, and at least one anti-oxidant inhibitor, wherein the perfusion produces impaired performance of the animal in memory and learning tests and induces abnormal neuropathology in a brain of the animal, wherein said impaired performance and abnormal neuropathology are in comparison with control non-human animals.

- 2. The non-human animal of claim 1, wherein the A β compound comprises A β_{42} .
- 3. The non-human animal of claim 1, wherein the A β compound comprises a peptide fragment of A β_{42} .
- 4. The non-human animal of claim 3, wherein the peptide fragment of $A\beta_{42}$ comprises at least one of $A\beta_{1-40}$ or $A\beta_{24-35}$.
- 5. The non-human animal of claim 1, wherein the $A\beta$ compound comprises a peptidomimetic that mimicks $A\beta_{42}$.
- 6. The non-human animal of claim 1, wherein the at least one pro-oxidative compound is selected from the group consisting of ferrous sulfate, copper sulfate, cobalt sulfate, manganese sulfate, and zinc sulfate.

- 7. The non-human animal of claim 1, wherein the at least one pro-oxidative compound comprises ferrous sulfate.
- 8. The non-human animal of claim 1, wherein the at least one anti-oxidant inhibitor comprises buthionine sulfoximine.
- 9. The non-human animal of claim 1, wherein the process further comprises perfusing the non-human animal with an effective amount of a phosphatase inhibitor.
- 10. The non-human animal of claim 9, wherein the phosphatase inhibitor is selected from the group consisting of okadaic acid, 1-nor-okadaone, bioallethrin, calycullin A, cantharidic acid, cantharidin, cypermethrin, deltamethrin, endothall, endothall thioanhydride, fenvalerate, okadol, permethrin, phenylarsine oxide, pyrophosphate, sodium fluoride, and vanadate.
- 11. The non-human animal of claim 9, wherein the phophatase inhibitor comprises okadaic acid.
- 12. The non-human animal of claim 1, wherein the process further comprises perfusing the non-human animal with an effective amount of a pro-inflammatory compound.
- 13. The non-human animal of claim 12, wherein the pro-inflammatory compound is selected from the group consisting of TNF-α, IL-6, and IL-1b.
- 14. The non-human animal of claim 12, wherein the pro-inflammatory compound comprises TNF- α .

15. A method for inducing a neurologic disease in a non-human animal, comprising:

perfusing the non-human animal with a pharmacologically effective amount of a combination of an $A\beta$ compound, at least one pro-oxidative compound, and at least one anti-oxidant inhibitor.

- 16. The method of claim 15, wherein the A β compound comprises A β_{42} .
- 17. The method of claim 15, wherein the A β compound comprises a peptide fragment of A β_{42} .
- 18. The method of claim 17, wherein the peptide fragment of $A\beta_{42}$ comprises at least one of $A\beta_{1-40}$ or $A\beta_{24-35}$.
- 19. The method of claim 15, wherein the A β compound comprises a peptidomimetic that mimicks A β ₄₂.
- 20. The method of claim 15, wherein the at least one pro-oxidative compound is selected from the group consisting of ferrous sulfate, copper sulfate, cobalt sulfate, manganese sulfate, and zinc sulfate.
- 21. The method of claim 15, wherein the at least one pro-oxidative compound comprises ferrous sulfate.
- 22. The method of claim 15, wherein the at least one anti-oxidant inhibitor comprises buthionine sulfoximine.

- 23. The method of Claim 15, further comprising perfusing the non-human animal with an effective amount of a phosphatase inhibitor.
- 24. The method of claim 23, wherein the phosphatase inhibitor is selected from the group consisting of okadaic acid, 1-nor-okadaone, bioallethrin, calycullin A, cantharidic acid, cantharidin, cypermethrin, deltamethrin, endothall, endothall thioanhydride, fenvalerte, okadol, permethrin, phenylarsine oxide, pyrophosphate, sodium fluoride, and vanadate.
- 25. The method of claim 23, wherein the phophatase inhibitor comprises okadaic acid.
- 26. The method of claim 15, further comprising perfusing the non-human animal with an effective amount of a pro-inflammatory compound.
- 27. The method of claim 27, wherein the pro-inflammatory compound is selected from the group consisting of TNF-α, IL-6, and IL-1b.
- 28. The method of claim 27, wherein the pro-inflammatory compound comprises TNF- α .
- 29. A method of screening for an agent that ameliorates symptoms of a neurologic disease, said method comprising:

comparing performance on memory and learning tests of a first non-human animal contacted with the agent with that of a second non-human animal not contacted with the agent, wherein the first and said second non-human animals have been co-

infused with a pharmacologically effective amount of $A\beta$, at least one pro-oxidative compound, and at least one anti-oxidant inhibitor wherein the co-infusion produces impaired performance on the memory and learning tests and abnormal neuropathology in a brain of the first and second non-human animals, wherein the impaired performance and the abnormal neuropathology are in comparison with control non-human animals, whereby an agent which ameliorates the symptoms is identified by superior performance of said first non-human animal in comparison with the second non-human animal on the memory and learning tests.

30. A method for screening for an agent useful for treating a neurologic disease, said method comprising:

comparing performance on memory and learning tests of a first non-human animal contacted with the agent with that of a second non-human animal not contacted with the agent, wherein the first and said second non-human animals have been coinfused with a pharmacologically effective amount of A\beta and at least one pro-oxidative compound, and at least one anti-oxidant inhibitor, wherein the co-infusion produces impaired performance on the memory and learning tests and abnormal neuropathology in a brain of the first and second non-human animals, wherein the impaired performance and the abnormal neuropathology are compared with control non-human animals; and comparing neuropathology in the brain of the first and the second non-human animal when said first non-human animal exhibits superior performance on the memory and learning tests compared with the second non-human animal, whereby an agent which is useful for treating a neurologic disease is identified by a decrease in neuropathologic findings in the first non-human animal in comparison with the second non-human animal.